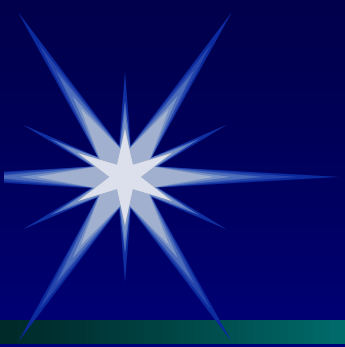


Dakahlia Nephrology Group
رابطة أطباء الكلى بالدقهلية

NMGH, Jan, 2014

Cancer and The Kidney
(Onco-Nephrology)

Hussein Sheashaa

Professor of Nephrology, Urology and Nephrology Center and Director of Medical E-Learning Unit, Mansoura University

Focus of The Talk

1- CKD and cancer

2- AKI and cancer

3- Malignancy associated glomerulopathy

4- Hypertension and cancer

5- Cancer related electrolyte and acid base disturbance

6- Radiation nephropathy

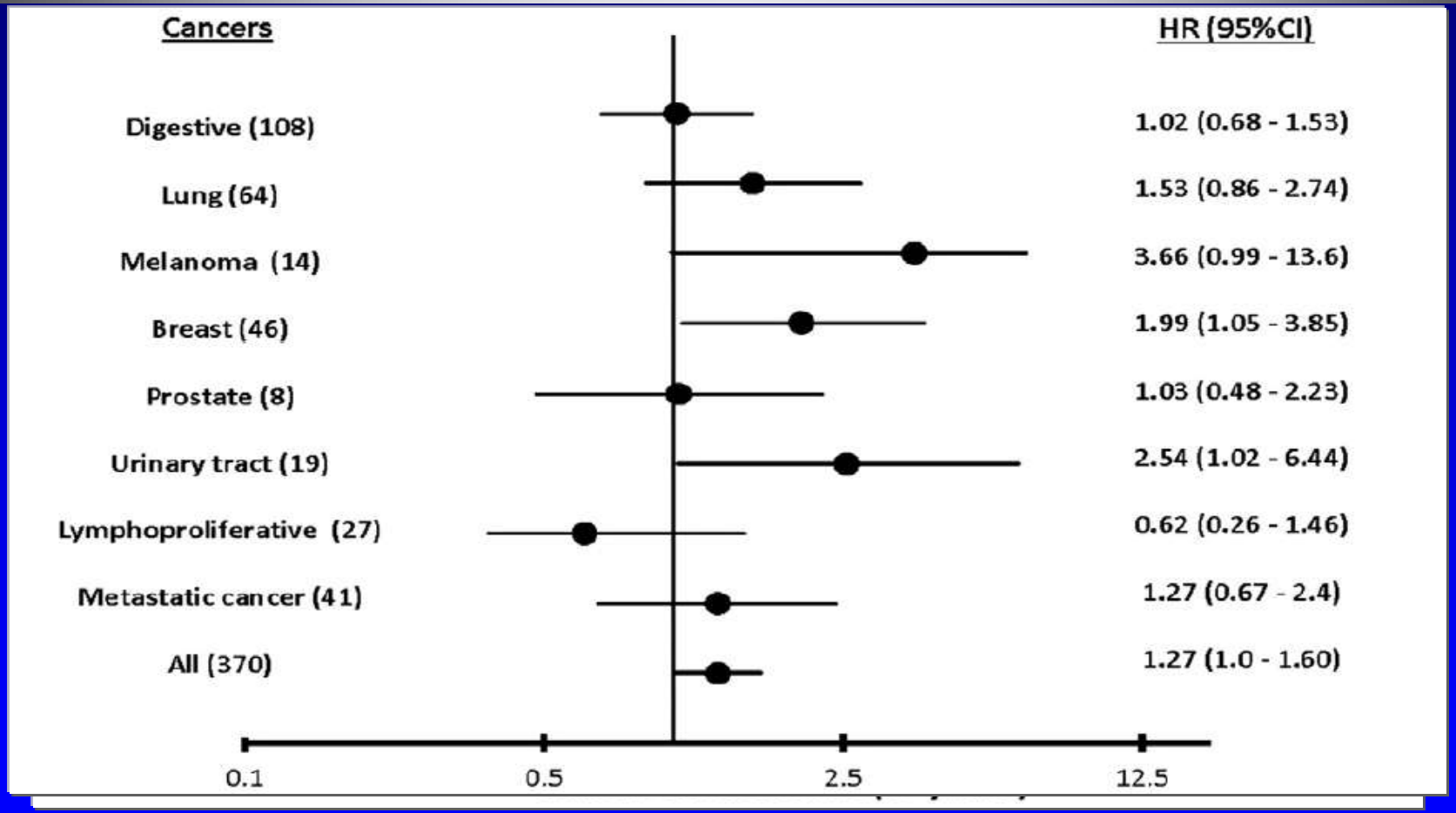
7- Post-transplant malignancy

8- Cases and teaching points

Awards

CKD and Cancer

CKD and Malignancy



Am J Kidney Dis. 2014;63(1):23-30

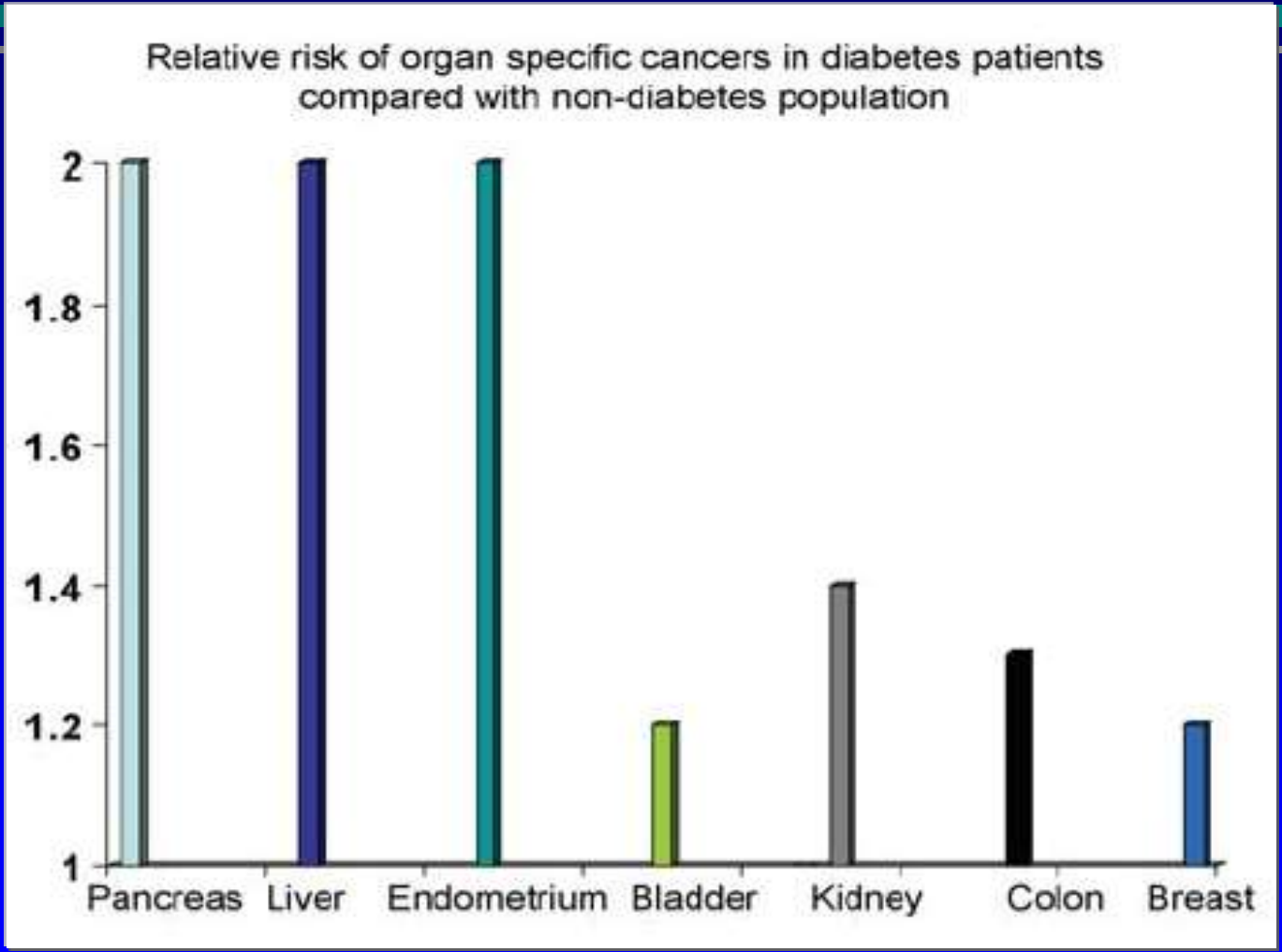
Cancer and CKD

Mechanisms by Which CKD Might Be Associated With Increased Risk of Cancer Death

Mechanism	Example	Comment
Death is independent of the cancer	Death from CV disease	CKD is a known risk factor for CV death
CKD or associated morbidities limit treatment options	Cisplatin	Concern re increased nephrotoxicity and other adverse effects; optimal dosing difficult
	Bisphosphonates in myeloma	Concern re increased nephrotoxicity; optimal dosing difficult
	Hematopoietic cell transplantation	Low GFR of itself not an absolute contraindication, but comorbid conditions are often present, making this aggressive treatment high risk

Am J Kidney Dis. 2014;63(1):7-9

DM and Malignancy



Nephrol Dial Transplant (2012) 27: 3018–3020

Malignancy in Lupus

- Malignancies develop in about 4.5% of lupus patients.
- The highest for non-Hodgkin's lymphomas, Hodgkin's disease, cervical, bronchial and breast cancers.
- Over-expression of B-cell activating factor.

Cancer and ESRD

Incidence of reported ESRD, by primary diagnosis, 2007–2011 combined

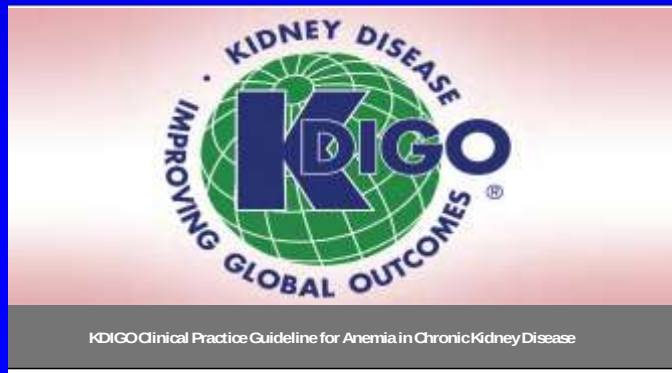
by detailed primary diagnosis

COLUMN PERCENT	Total patients	Counts White	Black/ Af Am	N Am	Asian	Counts Hisp.	Non-Hisp.	% White	Black/ Af Am	N Am	Asian	% Hisp.	Non-Hisp.
Neoplasms/tumors	11,311	8,933	2,075	71	223	857	10,454	3	1	1	1	1	2
Renal tumor (malignant)	2,278	1,882	352	16	28	147	2,131	1	0	0	0	0	0
Urinary tract tumor (malignant)	737	614	109	*	*	49	688	0	0	*	*	0	0
Renal tumor (benign)	87	70	12	.	*	*	81	0	0	.	*	*	0
Urinary tract tumor (benign)	49	47	*	.	.	*	44	0	*	.	.	*	0
Renal tumor (unspecified)	264	206	46	*	*	22	242	0	0	*	*	0	0
Urinary tract tumor (unspecified)	175	145	25	*	*	25	150	0	0	*	*	0	0
Lymphoma of kidneys	178	145	24	*	*	15	163	0	0	*	*	0	0
Multiple myeloma	5,482	4,136	1,188	33	121	410	5,072	1	1	1	1	1	1
Other immuno prolif. neoplasms (inc. light chain neph.)	693	557	112	*	19	41	652	0	0	*	0	0	0
Amyloidosis	1,368	1,131	205	*	23	137	1,231	0	0	*	0	0	0

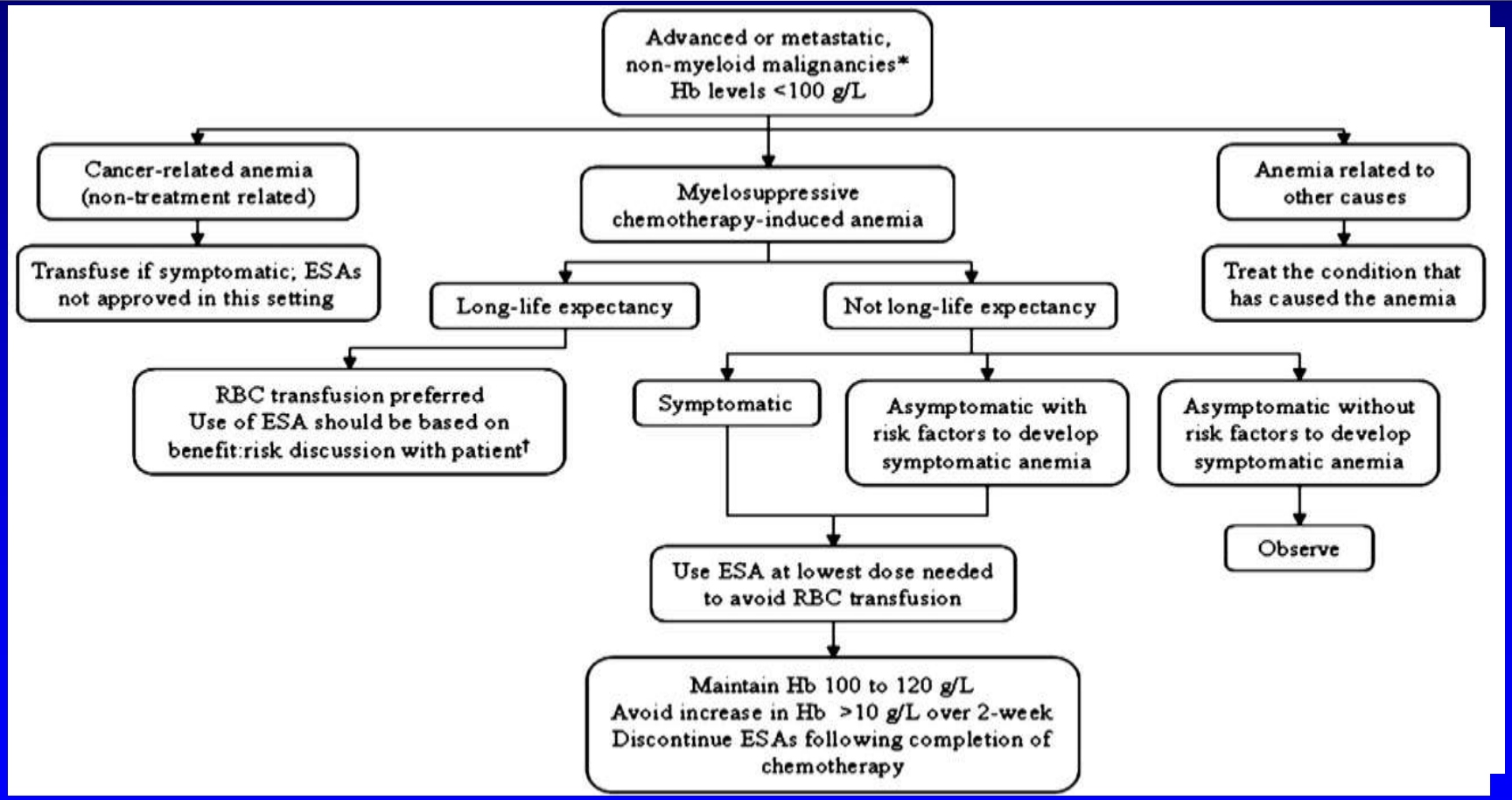
United States Renal Data System
2013 Annual Data Report

Cancer and CKD:
Anemia Management in CKD

3.3: We recommend using ESA therapy with great caution, if at all, in CKD patients with active malignancy—in particular when cure is the anticipated outcome—(*1B*), a history of stroke (*1B*), or a history of malignancy (*2C*).



Cancer Associated Anemia: ESA Therapy



Critical Reviews in Oncology/Hematology 87 (2013) 132–139

Cancer and CKD

4.4.8: People with CKD should not be denied therapies for other conditions such as cancer but there should be appropriate dose adjustment of cytotoxic drugs according to knowledge of GFR. (Not graded)

http://www.kidney-international.org review

Summary of KDIGO 2012 CKD Guideline: behind the scenes, need for guidance, and a framework for moving forward

Adeera Levin[1] and Paul E. Stevens[2]

Kidney International advance online publication, 27 November 2013;

[1]Division of Nephrology, University of British Columbia, Vancouver, British Columbia, Canada and [2]East Kent Hospitals University NHS Foundation Trust, Canterbury, UK

AKI and Cancer

Cancers with Highest AKI Risk

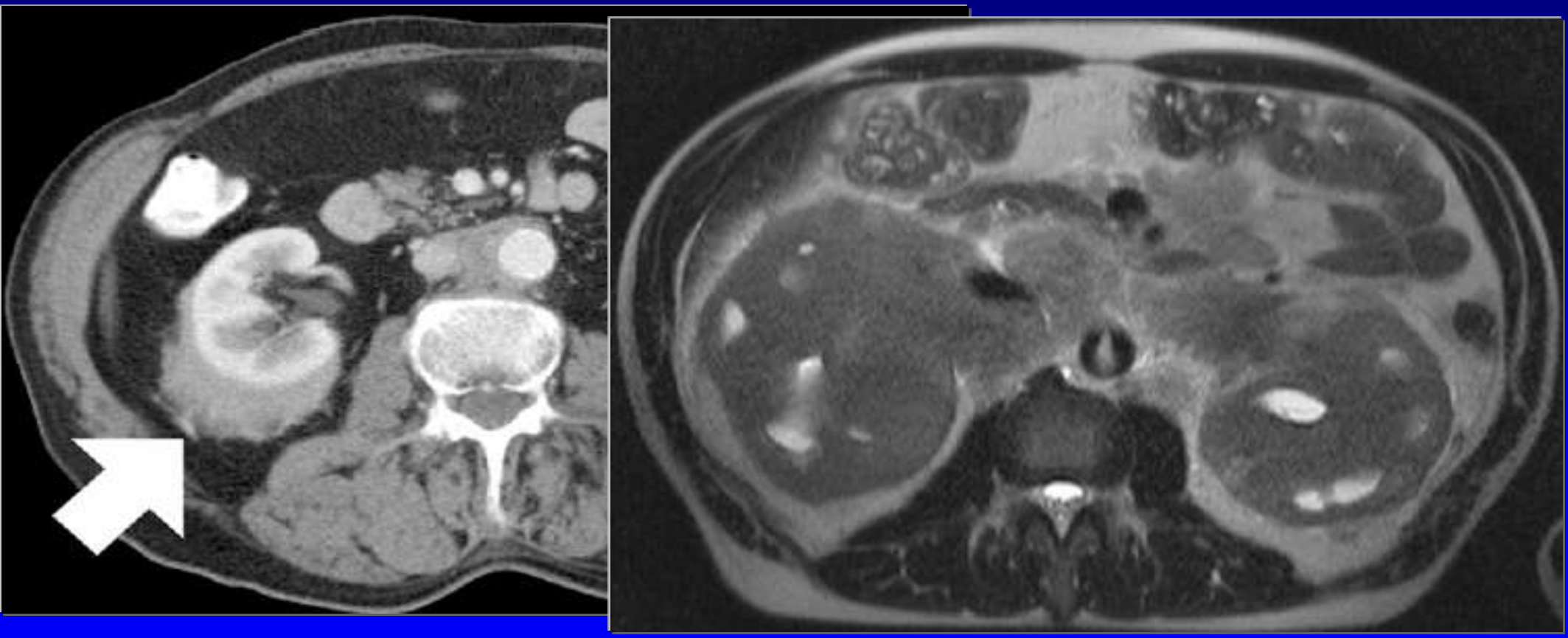
1. Kidney cancer
2. Multiple myeloma
3. Liver cancer
4. Acute lymphoma or leukemia

Renal Involvement in Lymphoma and Leukemia

1. Obstructive uropathy
2. Infiltration of renal parenchyma
3. Amyloidosis
4. Therapy associated
5. Urate nephropathy
6. Glomerulopathy
7. Disseminated intravenous coagulation

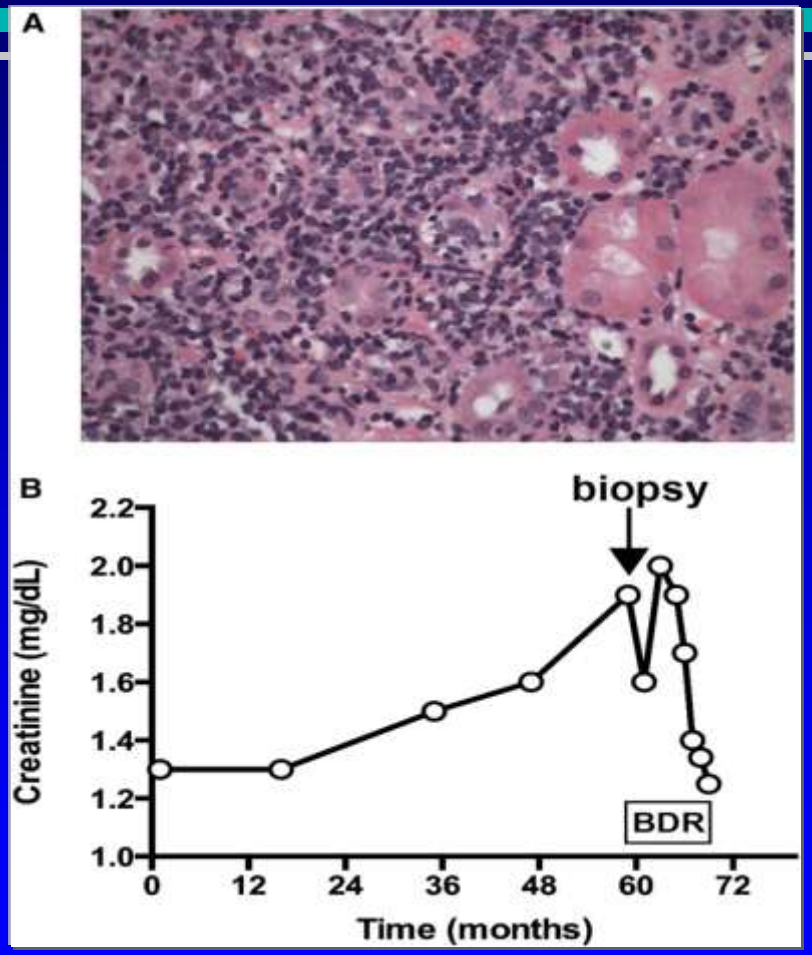
Lymphomatous Infiltration of The Kidneys (LIK)

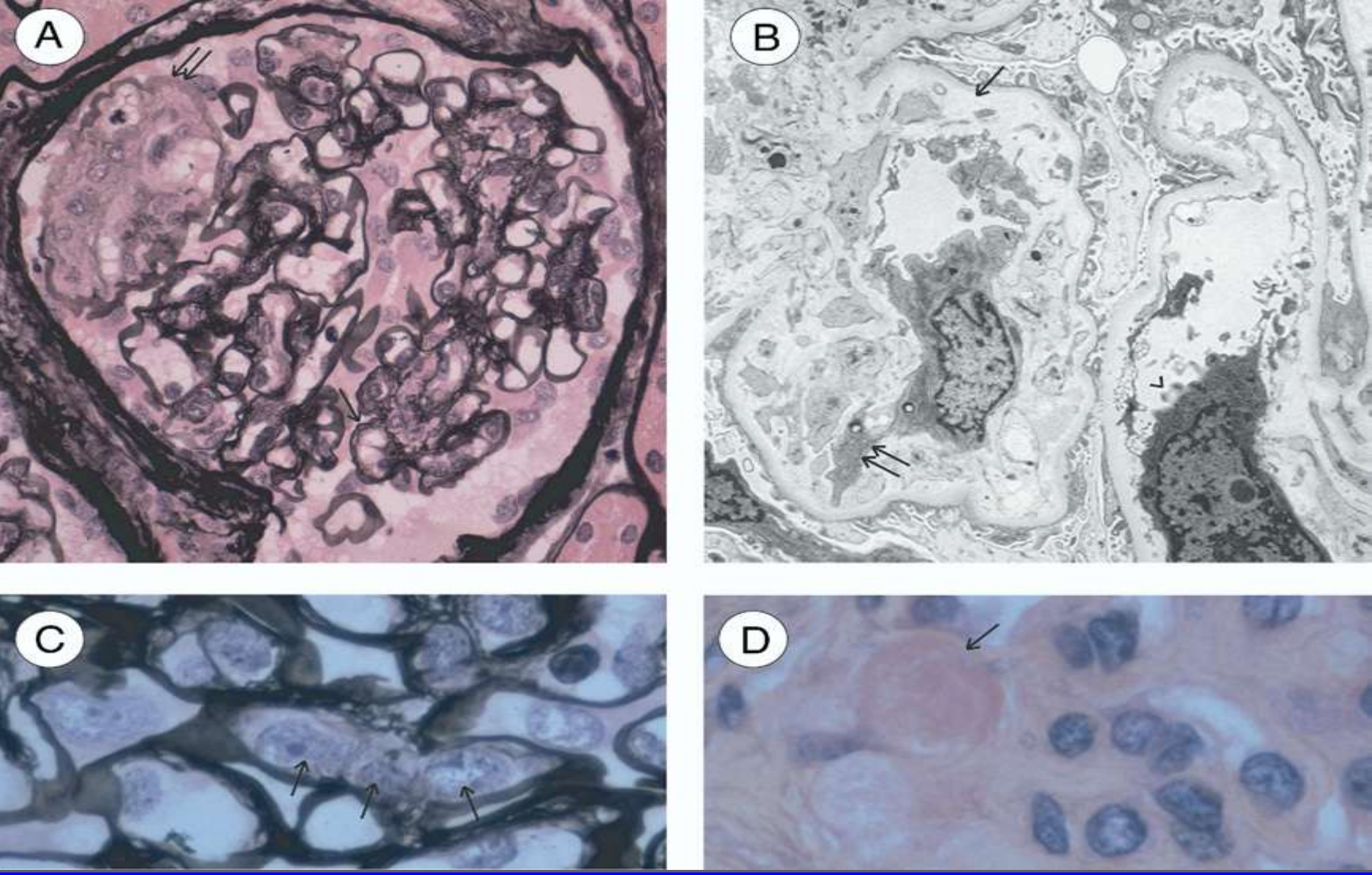
- **Mark true or false:**
  a) It is a rare disease
  b) Usually presents with AKI
  c) Most commonly, patients develop slowly progressive CKD
  d) LIK is an indication of chemotherapy



LIK

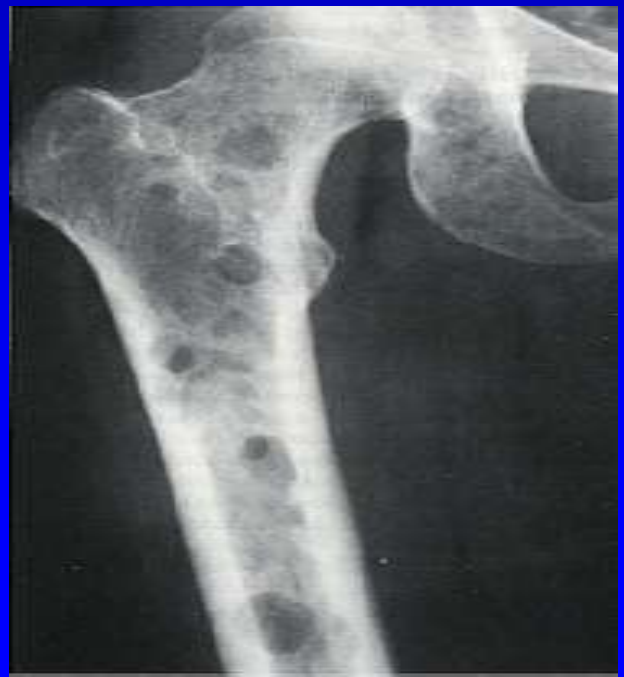
BDR; bortezomib, dexamethasone, rituxan

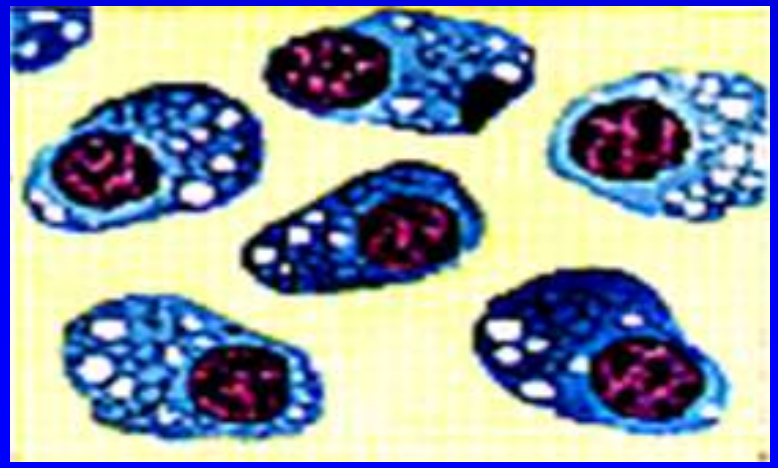




American Journal of Kidney Diseases, Vol 53, No 3 (March), 2009: pp 550-554

Renal Involvement in Multiple Myeloma





Multiple Myeloma and AKI

Mechanisms of renal failure in plasma cell dyscrasias: Ig-dependent and -independent

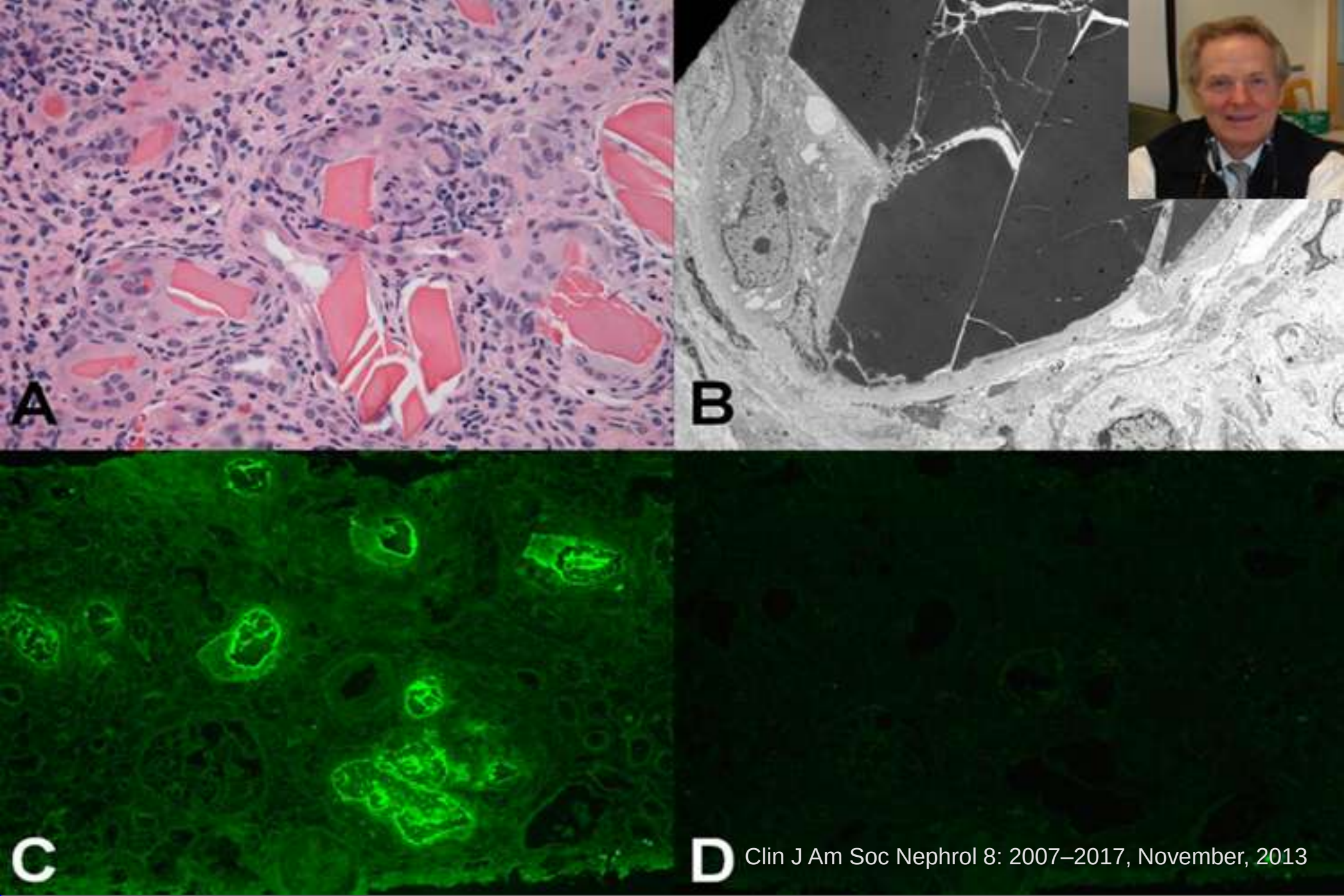
Mechanism	Details
Ig-independent mechanisms	
Volume depletion	Can cause prerenal azotemia and acute tubular necrosis and/or contribute to cast nephropathy
Sepsis	
Hypercalcemia	Can cause AKI directly or contribute to cast nephropathy
Tumor lysis syndrome	Uric acid or phosphate nephropathy
Medication toxicity	Zoledronate: rare cause of acute renal failure
	Pamidronate: rare cause of collapsing focal and segmental glomerulosclerosis
	Nonsteriodal anti-inflammatory drugs, angiotensin converting enzyme inhibitor, angiotensin receptor blocker, loop diuretics, or iodinated contrast may precipitate cast nephropathy
Direct parenchymal invasion by plasma cells	Rare cause; associated with advanced or aggressive myeloma
Pyelonephritis	Rare cause; multifactorial from immunodeficiency and deficient Ig and chemotherapy from myeloma

Clin J Am Soc Nephrol 8: 2007–2017, November, 2013

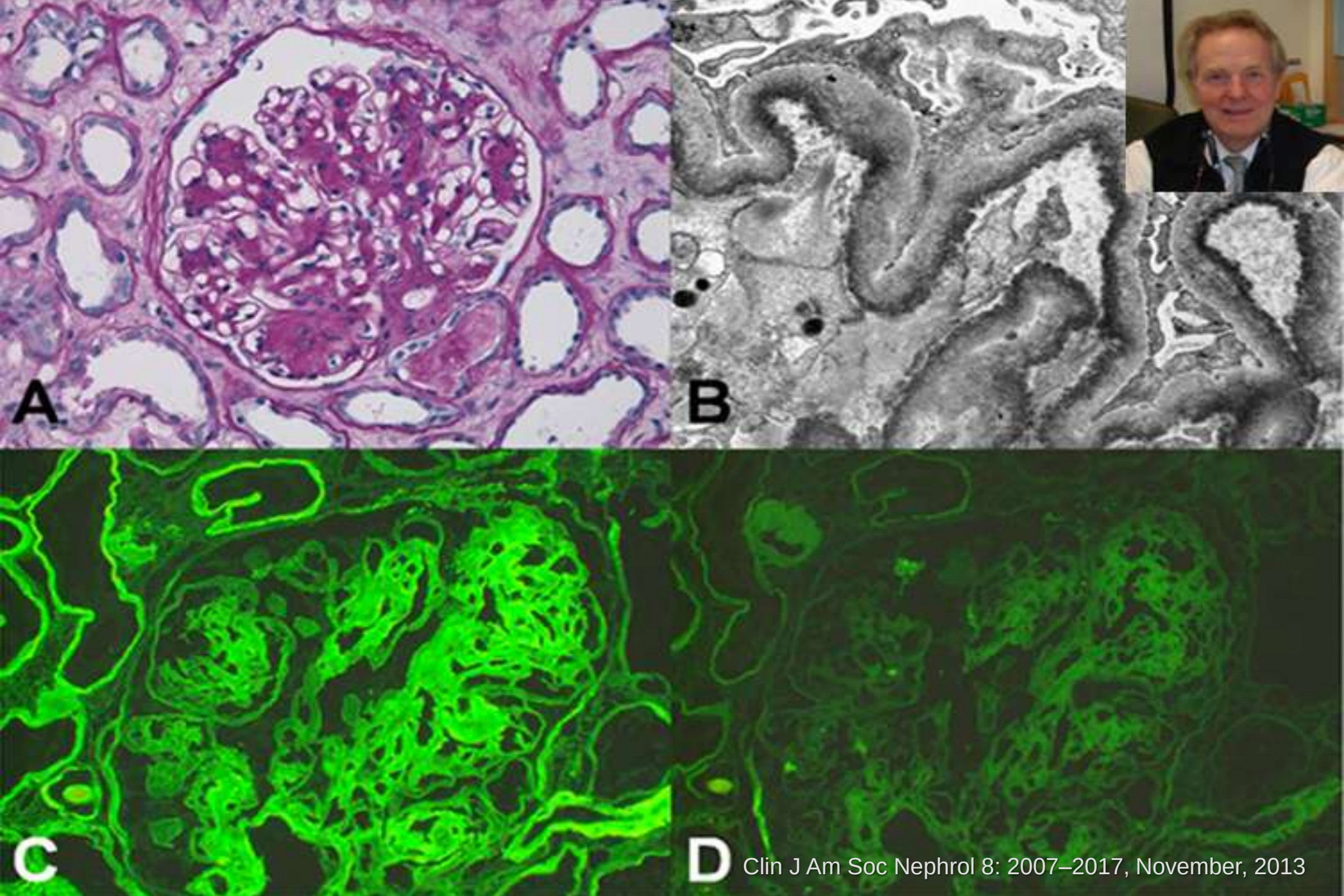
Renal Pathology in Patients with Multiple Myeloma

Histological Finding	Prevalence
Myeloma kidney *(Myeloma cast nephropathy)*	30%-50%
Interstitial nephritis/fibrosis without cast nephropathy	20%-30%
Amyloidosis	10%
Light chain deposition disease	5%
Acute tubular necrosis	10%
Other (urate nephropathy, tubular crystals, hypercalcemia, FSGS)	5%

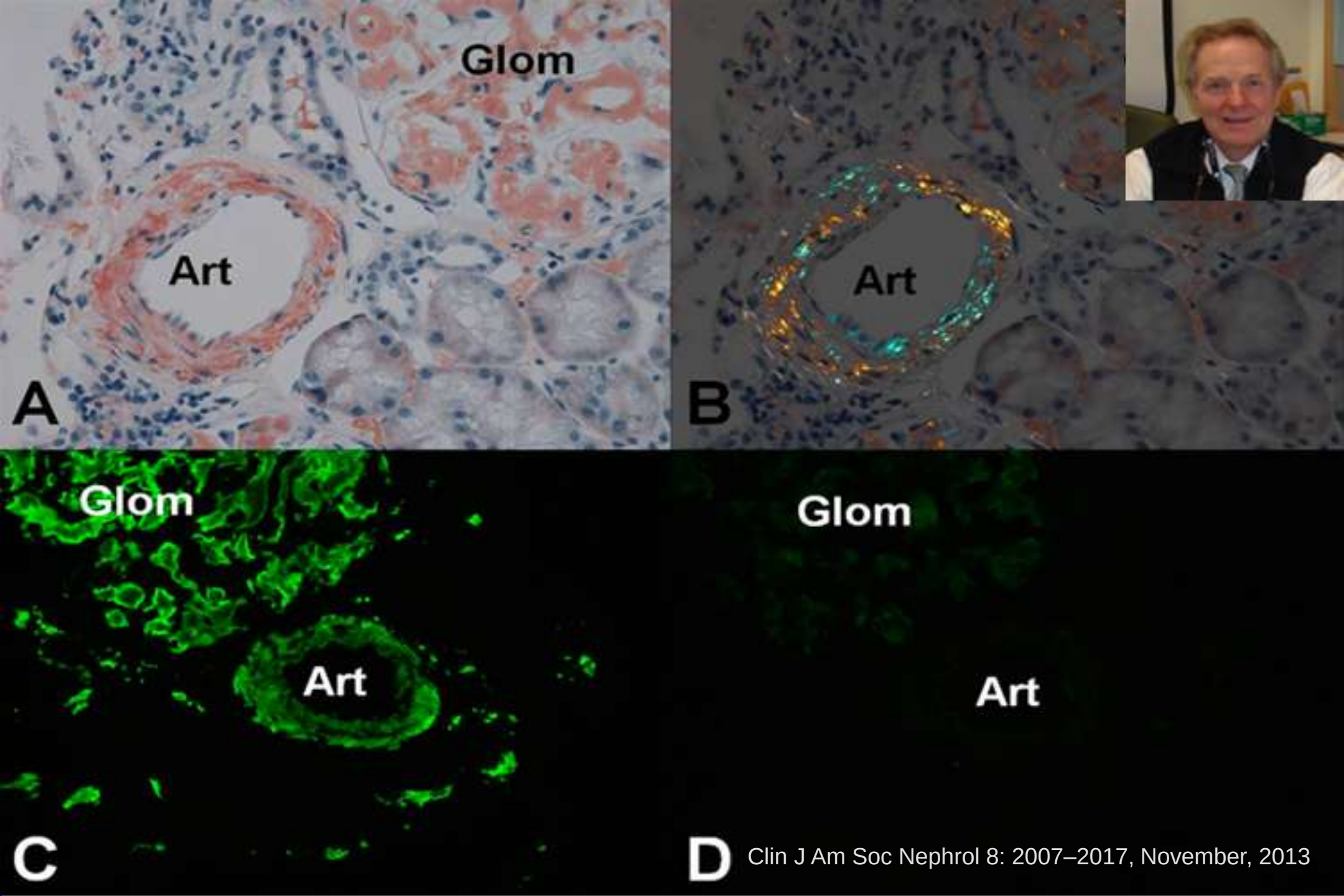
Comprehensive textbook of Nephrology, 2010 ed



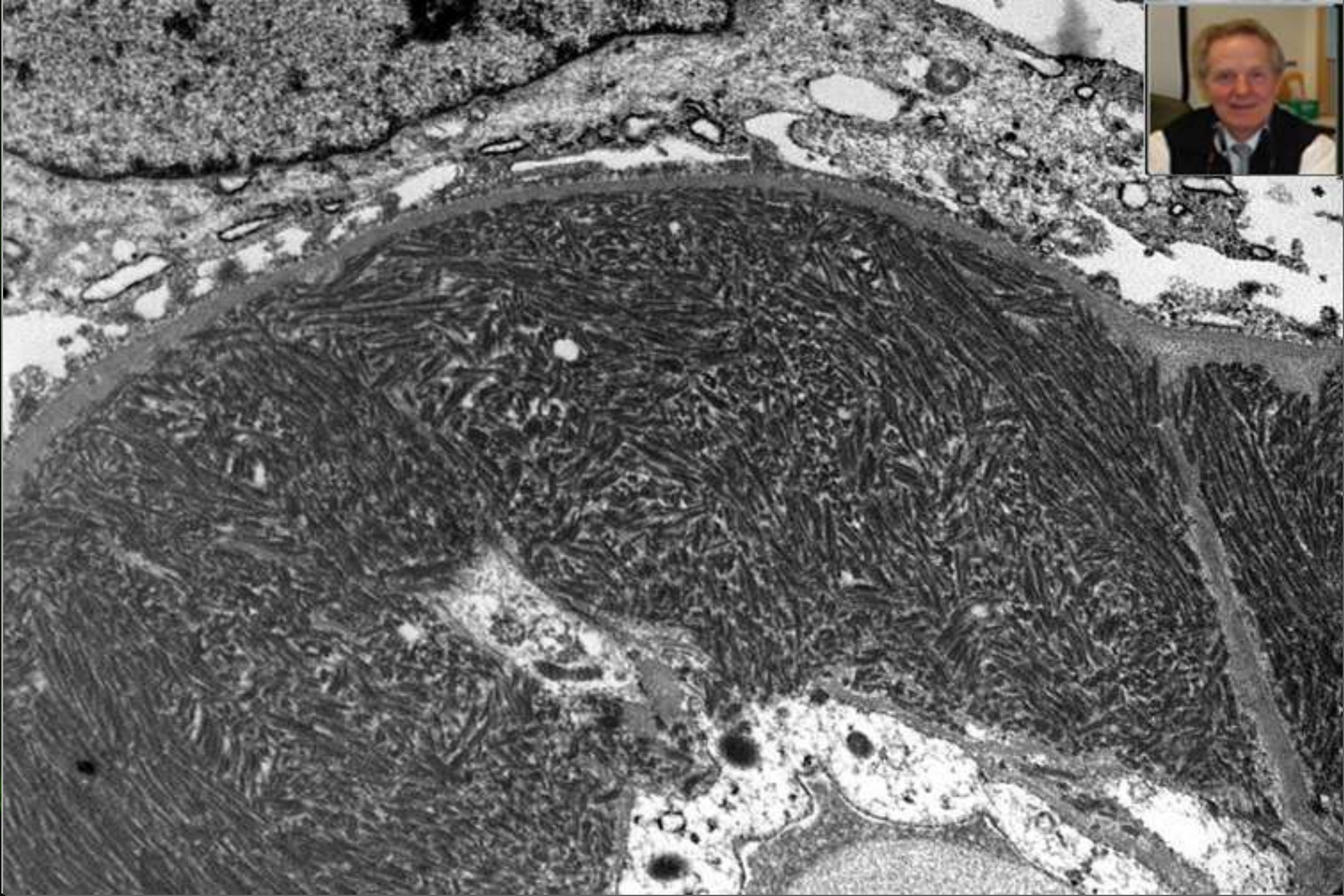
Clin J Am Soc Nephrol 8: 2007–2017, November, 2013



Clin J Am Soc Nephrol 8: 2007–2017, November, 2013



Clin J Am Soc Nephrol 8: 2007–2017, November, 2013



Theralite™

High Cut-off technology

DOI 10.5414/CN107357

Clinical Nephrology, Volume 79 - April (318 - 322)

Recovery of kidney function following delayed use of theralite™ dialyzer in a patient with myeloma cast nephropathy

Khagendra Dahal[1], Shani Shastri[1], Uma Narayanasami[2, 3], Vanesa Bijol[4], Karen Rider[1], James A. Strom[1, 3], Bertrand L. Jaber[1, 3]

[1] Division of Nephrology, [2] Division of Hematology/Oncology, Department of Medicine, St. Elizabeth's Medical Center,
[3] Department of Medicine, Tufts University, School of Medicine,
[4] Department of Pathology, Brigham, Women's Hospital, Boston, MA, USA

Clin Nephrol. 2013 Apr;79(4):318-22.

Treatment of Acute Renal Failure Secondary to Multiple Myeloma with Chemotherapy and Extended High Cut-Off Hemodialysis

Colin A. Hutchison,*† Arthur R. Bradwell,‡ Mark Cook,§ Kolitha Basnayake,*† Supratik Basu,‖ Stephen Harding,¶ John Hattersley,** Neil D. Evans,** Mike J. Chappel,** Paul Sampson,* Lukas Foggensteiner,* Dwomoa Adu,* and Paul Cockwell*†

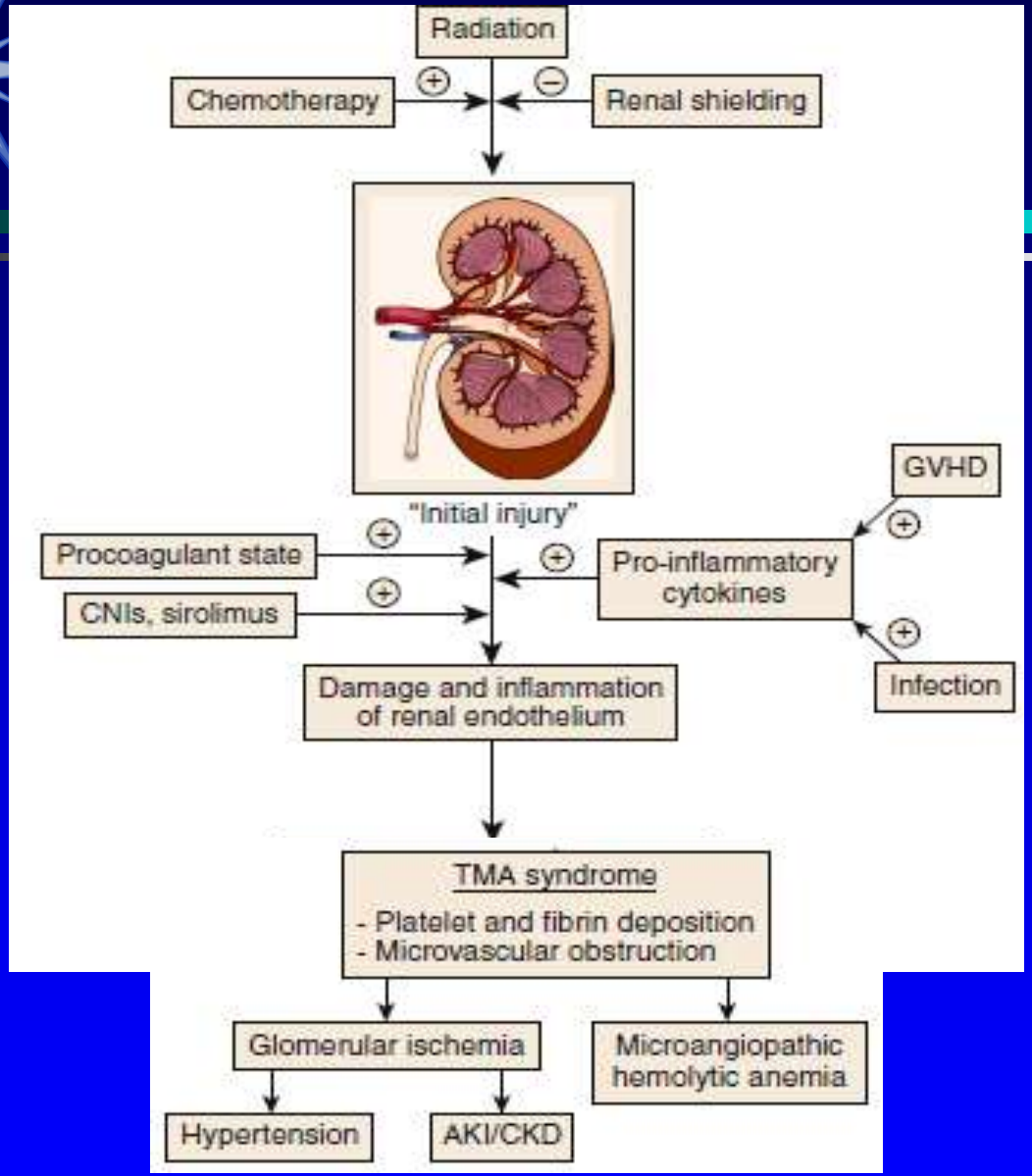
**Department of Nephrology and §Department of Haematology, Queen Elizabeth Hospital, Birmingham; †Division of Medical Sciences and ‡Division of Immunity and Infection, Medical School, University of Birmingham; ‖Department of Haematology, New Cross Hospital, Wolverhampton; ¶The Binding Site Ltd., Birmingham; **School of Engineering, University of Warwick, Coventry, United Kingdom*

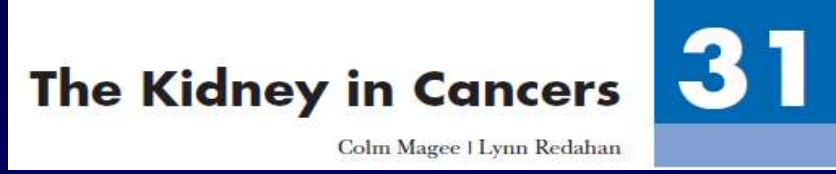
Clin J Am Soc Nephrol 4: 745–754, 2009.

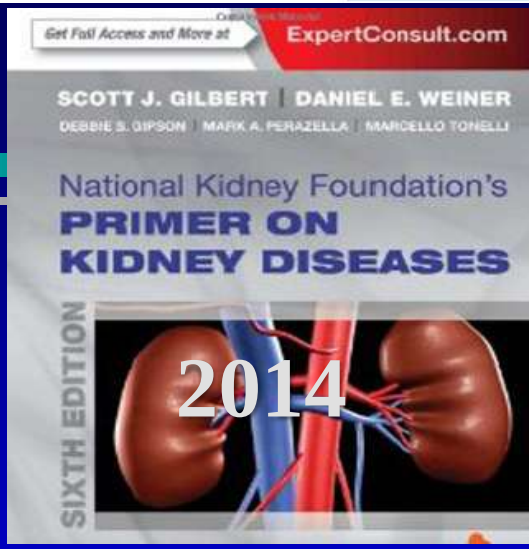
Causes of AKI After Hematopoietic Cell Transplant

- Early onset (<30 days)
  - Sepsis
  - Hypotension
  - Hypovolemia (vomiting and diarrhea)
  - Nephrotoxic agents
    - Acyclovir
    - Allopurinol
    - Amphotericin B
    - Angiotensin-converting enzyme inhibitors
    - Angiotensin receptor blockers
    - Calcineurin inhibitors
    - Contrast dye
    - Methotrexate
    - NSAIDs
  - Tumor lysis syndrome
  - Hepatic sinusoidal obstruction syndrome
- Late onset (>3 months)
  - Thrombotic microangiopathy
  - Calcineurin inhibitor toxicity

Clin J Am Soc Nephrol 7: 1692–1700, October, 2012


Radiation
Chemotherapy
Renal shielding
"Initial injury"
GVHD
Procoagulant state
CNIs, sirolimus
Pro-inflammatory cytokines
Infection
Damage and inflammation of renal endothelium
TMA syndrome
- Platelet and fibrin deposition
- Microvascular obstruction
Glomerular ischemia
Microangiopathic hemolytic anemia
Hypertension
AKI/CKD


The Kidney in Cancers
31
Colm Magee | Lynn Redahan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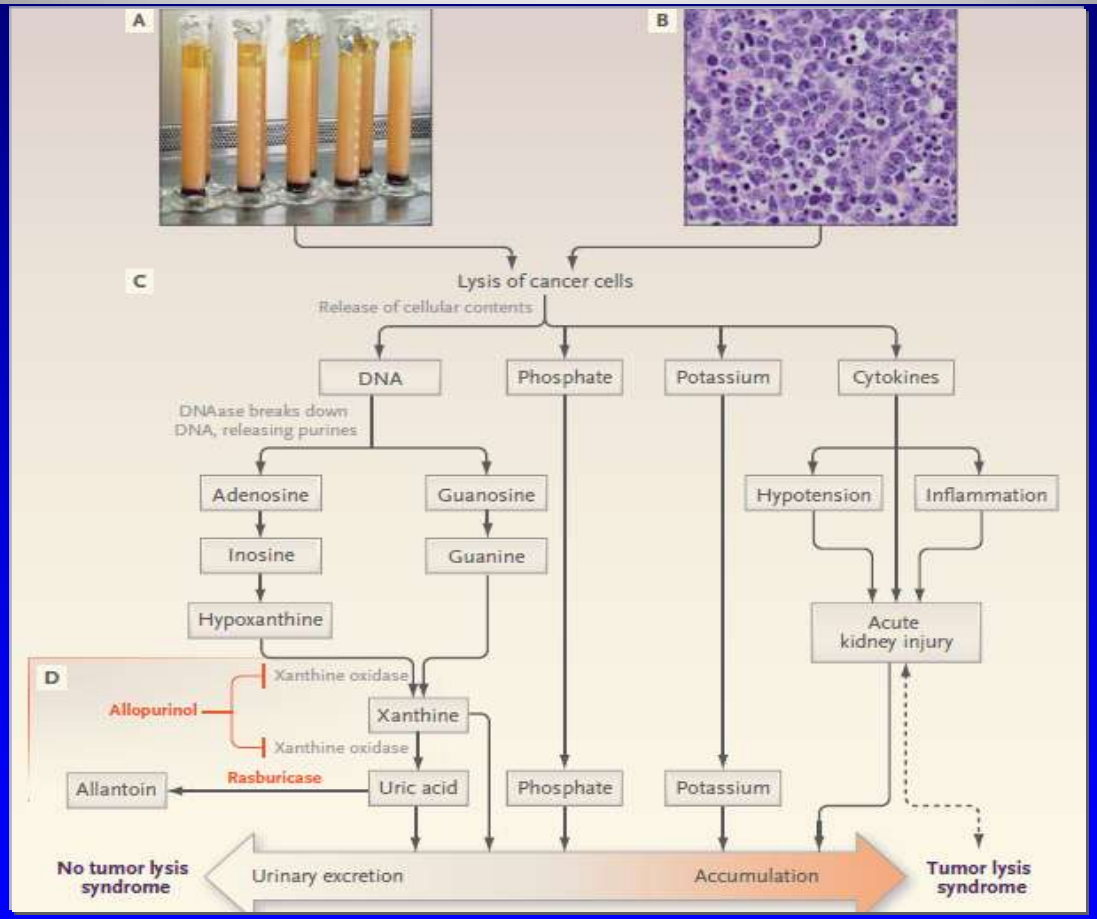
National Kidney Foundation's
PRIMER ON
KIDNEY DISEASES
SIXTH EDITION
2014

Treatment of HCT Associated TMA

- **Which is wrong in treating this complication?**
  a) BP control to below 140/80 mmHg
  b) Plasmapheresis is the treatment of choice
  c) Loop diuretic therapy
  d) Angiotensin converting enzyme inhibition
  e) Discontinuation of NSAID

TLS



N Engl J Med 2011;364:1844-54.

TLS

Definitions of laboratory TLS and clinical TLS proposed by Cairo and Bishop and modified by Howard *et al.*

Laboratory TLS	Clinical TLS
(Requires ≥2 laboratory abnormalities)	(Requires laboratory TLS features plus any clinical finding below)
Hyperuricemia (uric acid ≥8 mg/dl)	AKI≥
Hyperphosphatemia (>4.5 mg/dl in adults; >6.5 mg/dl in children)	Stage I (AKIN criteria)≥ R (RIFLE criteria)
Hyperkalemia (potassium >6.0 mEq/L)	Cardiac dysrhythmia, sudden death
Hypocalcemia (corrected serum calcium <7.0 mg/dl, or ionized calcium <1.12 mg/dl)	Cardiac dysrhythmia, sudden death, seizure, tetany, carpopedal spasm, bronchospasm, laryngospasm, hypotension

AKIN, Acute Kidney Injury Network. Data are from the following studies: Cairo MS, Bishop M: Tumour lysis syndrome: New therapeutic strategies and classification. *Br J Haematol* 127: 3–11, 2004; and from Howard SC, Jones DP, Pui CH: The tumor lysis syndrome. *N Engl J Med* 364: 1844–1854, 2011.

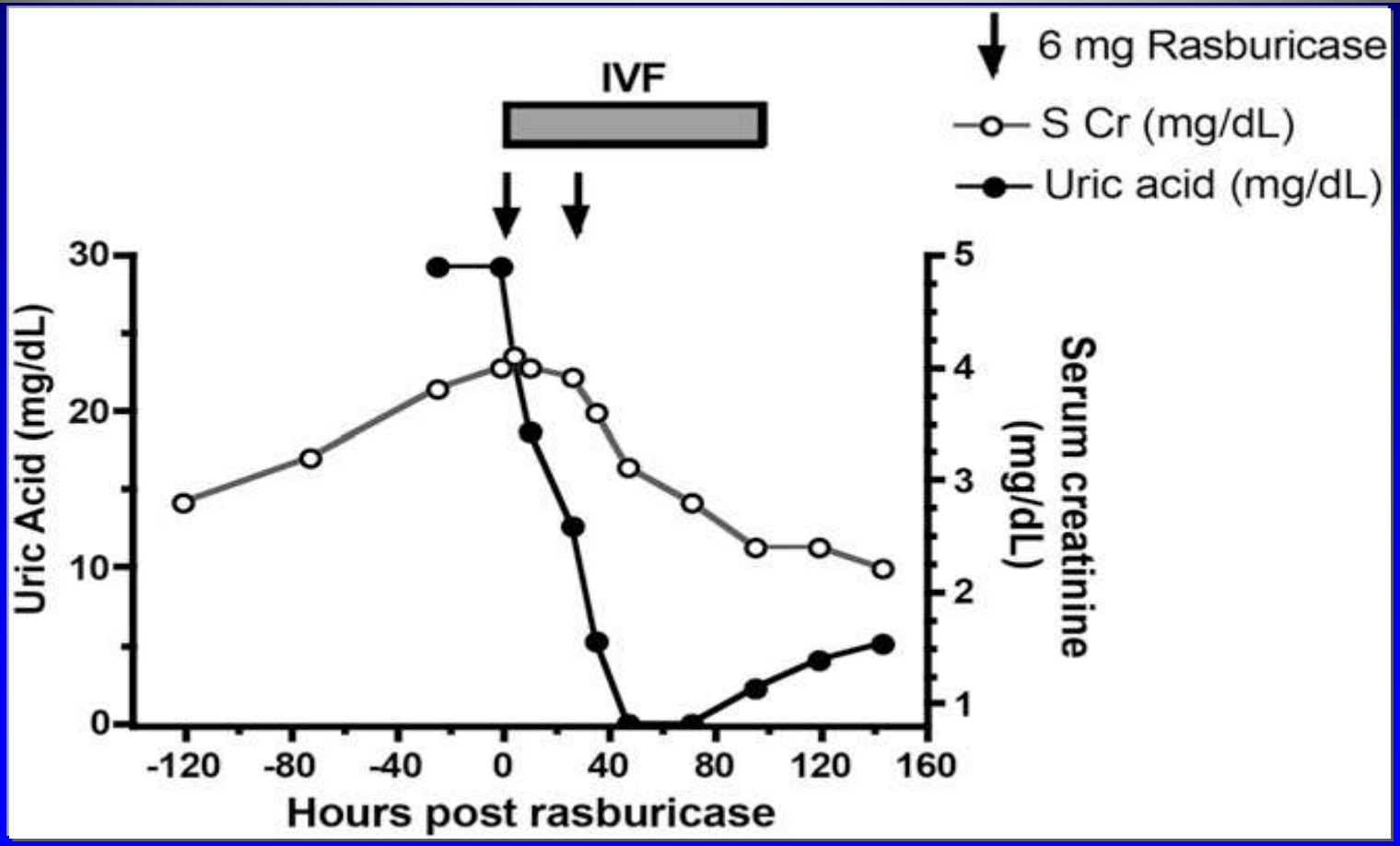
TLS

Risk factors that predispose to TLS

Risk Factor	Description
Tumor mass	Large tumor mass, extensive metastases
	Organ (kidney, liver, bone marrow) infiltration
	High rate of cell proliferation: LDH, WBC count, etc.
	Cancer cell type: hematological versus nonhematological
Acute cell lysis potential	Chemosensitivity
	Intensity of chemotherapy
Underlying conditions	Underlying CKD
	Hypotension
	Volume depletion
	Nephrotoxin exposure

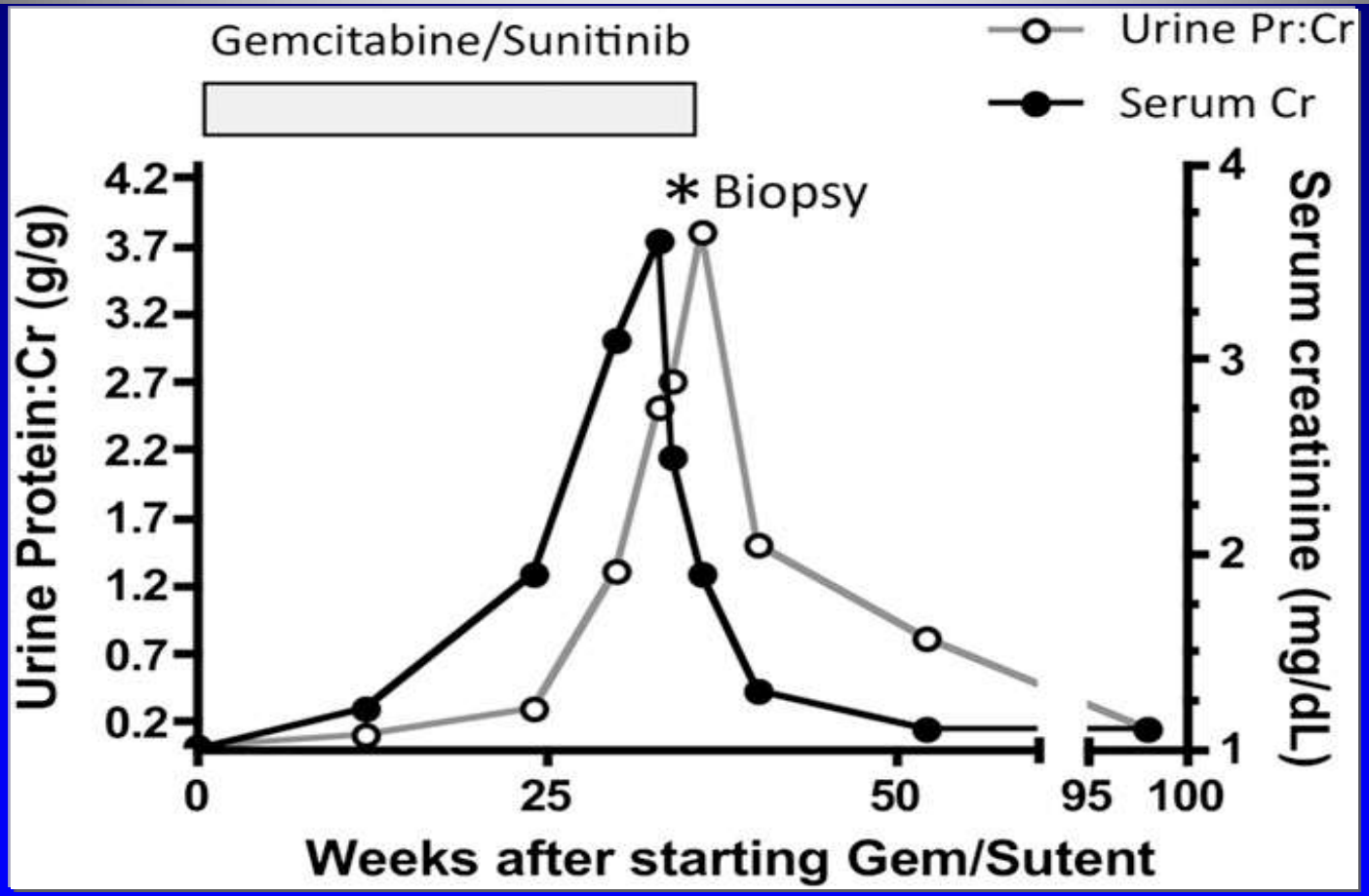
Nephrology Self-Assessment Program - Vol 12, No 1, January 2013

TLS



Nephrology Self-Assessment Program - Vol 12, No 1, January 2013

A 39Y-old woman with RCC



Nephrology Self-Assessment Program - Vol 12, No 1, January 2013

review

http://www.kidney-international.org

Glomerular diseases seen with cancer and chemotherapy: a narrative review

Kenar D. Jhaveri[1], Hitesh H. Shah[1], Kellie Calderon[1], Eric S. Campenot[2] and Jai Radhakrishnan[3]

[1]Division of Kidney Diseases and Hypertension, North Shore University Hospital and Long Island Jewish Medical Center, Hofstra North Shore-LIJ School of Medicine, Great Neck, New York, USA; [2]Department of Pathology and Cell Biology, Columbia University Medical Center, Columbia University College of Physicians and Surgeons, New York, New York, USA and [3]Division of Nephrology, Columbia University Medical Center, Columbia University College of Physicians and Surgeons, New York, New York, USA

Kidney International (2013) 84, 34–44;

Malignancy Associated Glomerulopathy

Endothelial damage (TMA)

Mitomycin C, gemcitabine, anti-VEGF agents, TKI, mTOR inhibitors, calcineurin inhibitors

Epithelial (podocyte) damage

Collapsing FSGS: pamidronate, mTOR inhibitors, calcineurin inhibitors, interferons α, β, and γ, adriamycin

FSGS NOS: interferons α, β, and γ, calcineurin inhibitors, mTOR inhibitors, daunorubicin

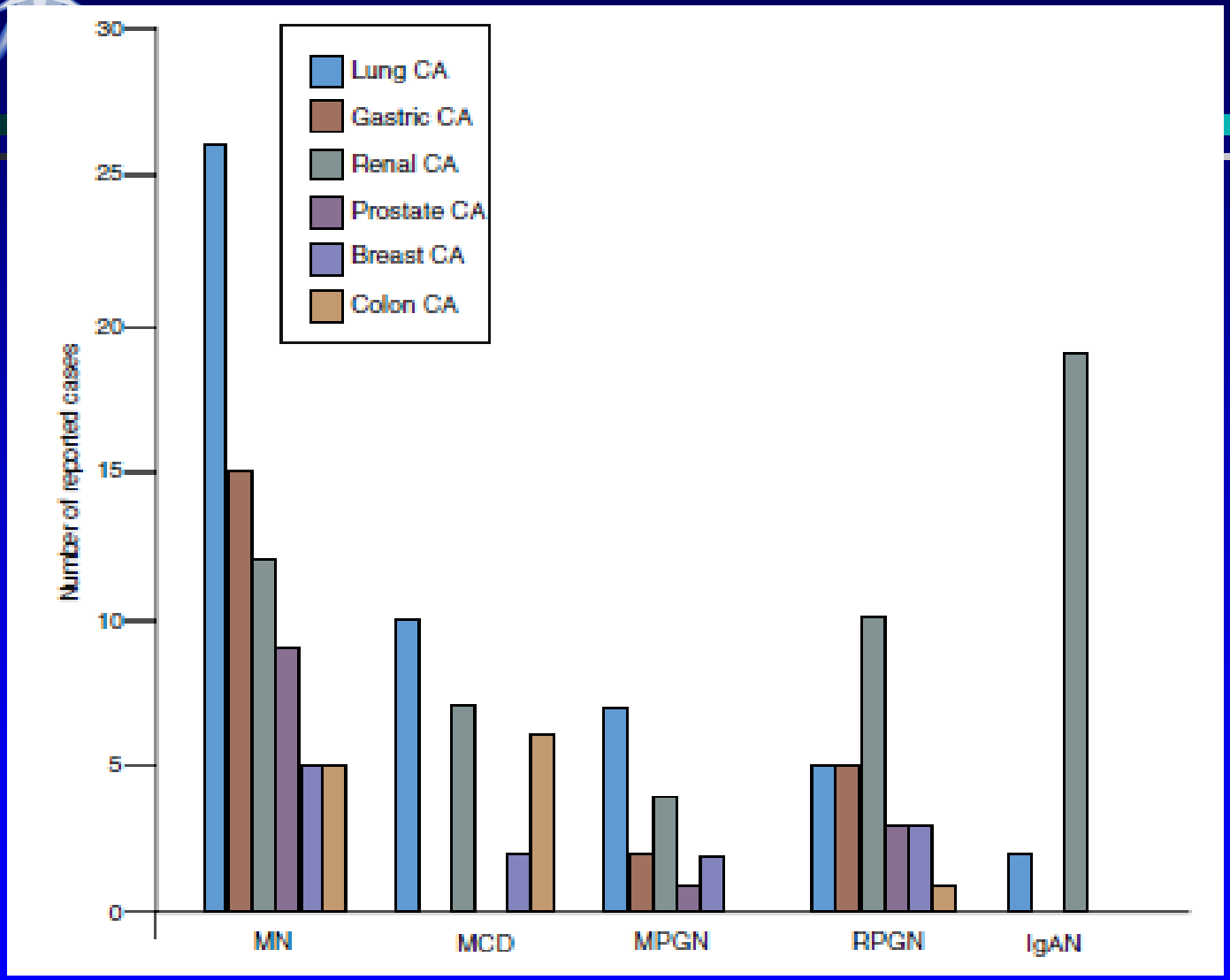
Minimal change disease: pamidronate, interferons α, β, and γ, daunorubicin

MPGN: anti-VEGF agents

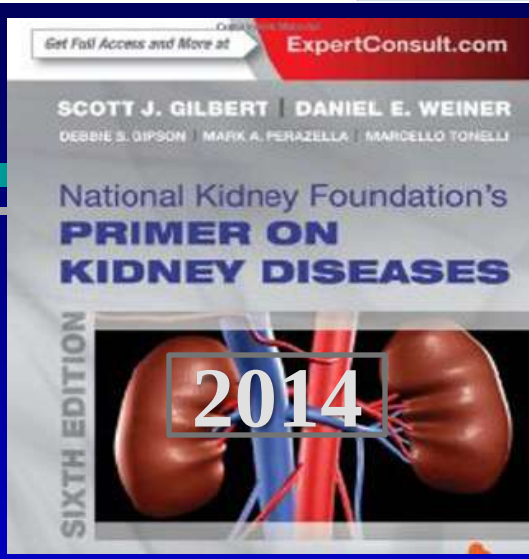
Crescentic GN: GM-CSF

Lupus-like nephritis: ipilimumab

Kidney International (2013) 84, 34–44;



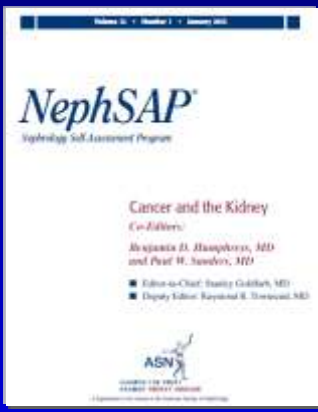
Nat Rev Nephrol 7:85-95, 2011.



Hypertension and Cancer

Hypertension Induced by Antiangiogenic Therapies

Which is true?



a) The mechanisms of hypertensive syndrome are not similar to preeclampsia.

b) Hypertension occurs in 5% of patients receiving antiangiogenic therapies.

c) Patients that develop hypertension on antiangiogenic therapies may have a superior antitumor response.

d) Discontinue herapy if the patient needs 3 antihypertensives.

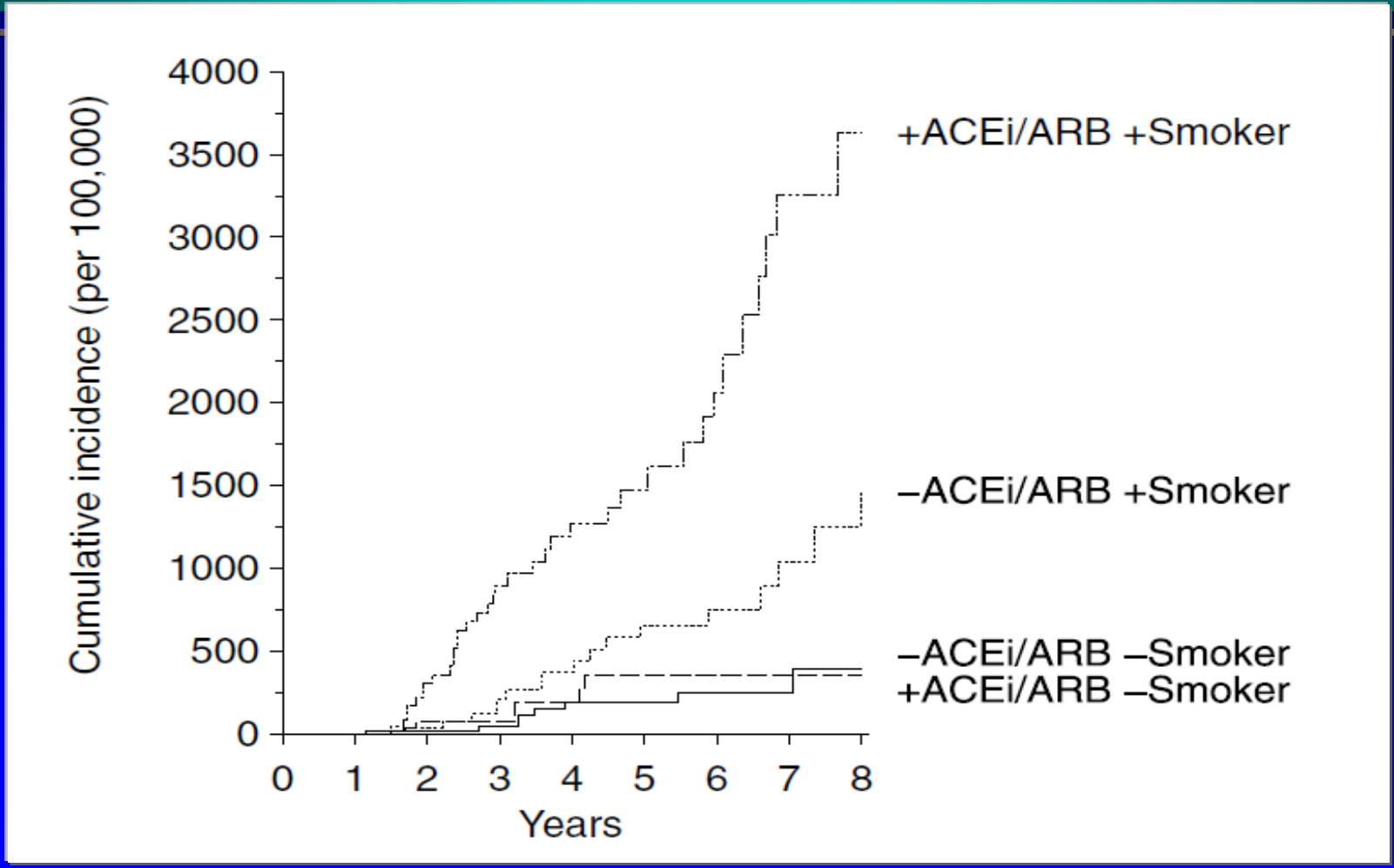
Antihypertensive Therapy and Cancer

American Journal of Transplantation 2011; 11: 2483–2489
Wiley Periodicals Inc.

doi: 10.1111/j.1600-6143.2011.03681.x

Treatment of Kidney Transplant Recipients With ACEi/ARB and Risk of Respiratory Tract Cancer: A Collaborative Transplant Study Report

Antihypertensive Therapy and Cancer



American Journal of Transplantation 2011; 11: 2483–2489

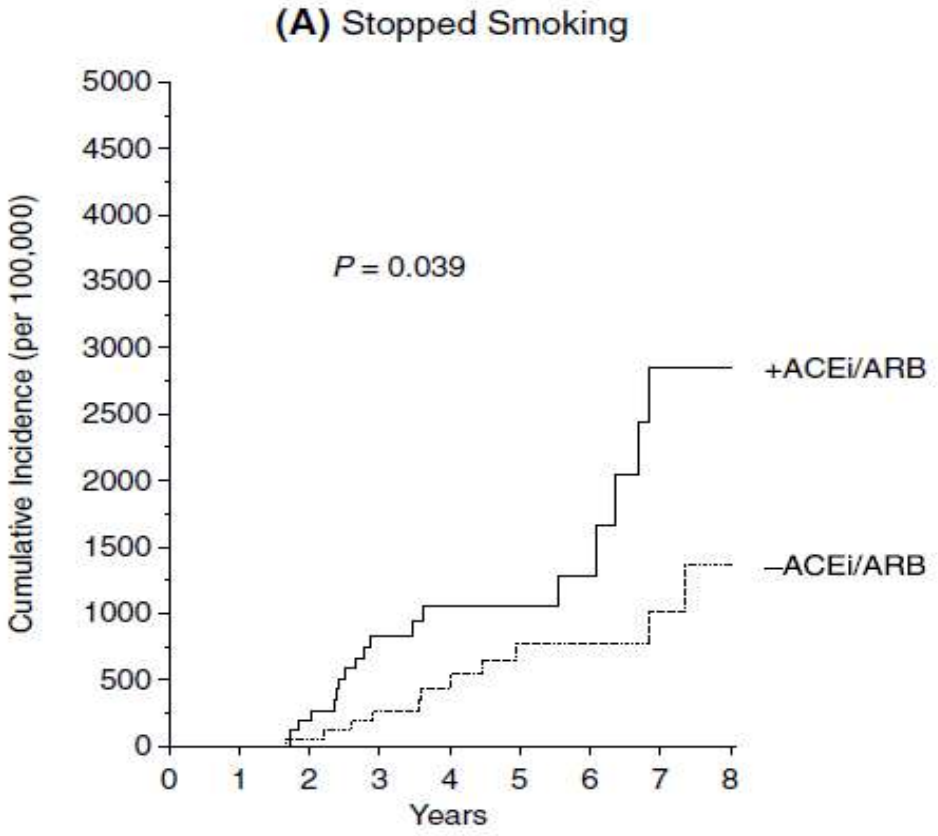
Antihypertensive Therapy and Cancer

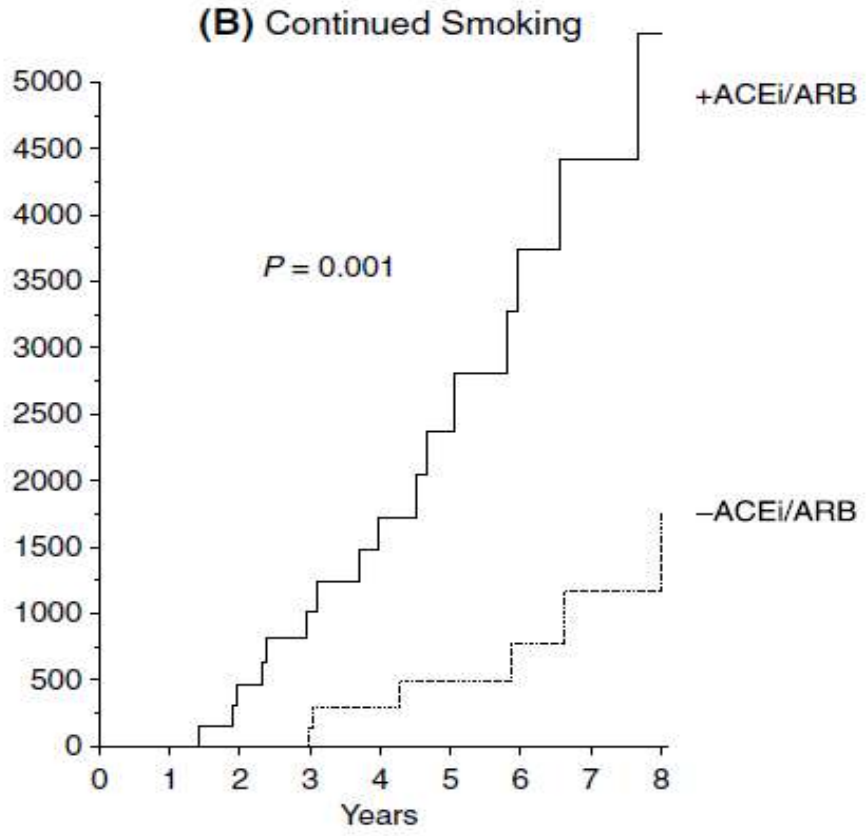
Significant confounders in Cox regression of malignant tumors occurring during posttransplant years 2–8

Confounder	HR	95% CI	p-Value
ACEi/ARB, history of smoking			
–ACEi/ARB –Smoker	1 (reference)		
–ACEi/ARB +Smoker	1.27	0.99–1.62	0.056
+ACEi/ARB –Smoker	1.09	0.84–1.41	0.53
+ACEi/ARB +Smoker	1.64	1.28–2.09	< 0.001

American Journal of Transplantation 2011; 11: 2483–2489

Antihypertensive Therapy and Cancer





American Journal of Transplantation 2011; 11: 2483–2489

Antihypertensive Therapy and Cancer

AJKD

In the Literature

Angiotensin II Receptor Blockers and Risk of Cancer: Cause for Concern?

Commentary on Sipahi I, Debanne SM, Rowland DY, Simon DI, Fang JC. Angiotensin-receptor blockade and risk of cancer: meta-analysis of randomised controlled trials. Lancet Oncol. 2010;11(7):627-636.

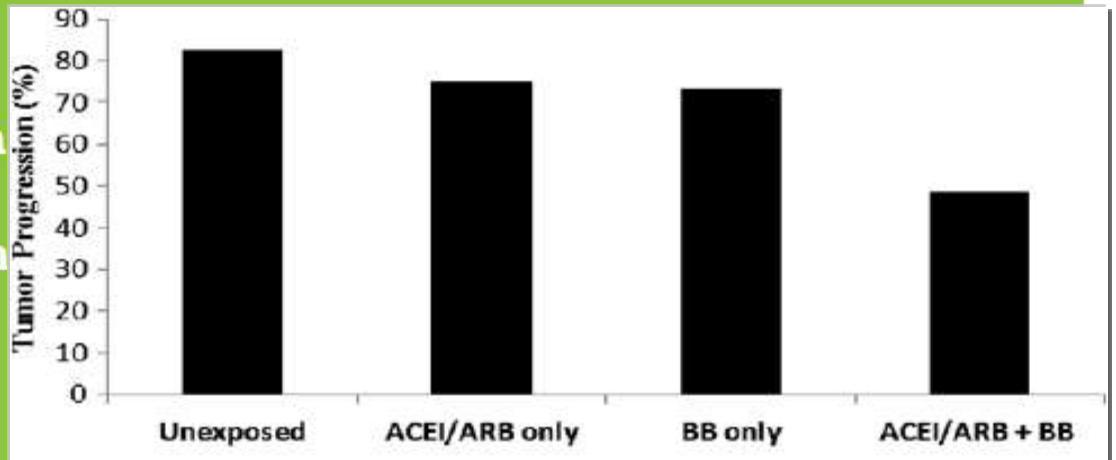
Am J Kidney Dis. 2011;57(1):7-10

Antihypertensive Therapy and Cancer

Translational Oncology
www.transonc.com

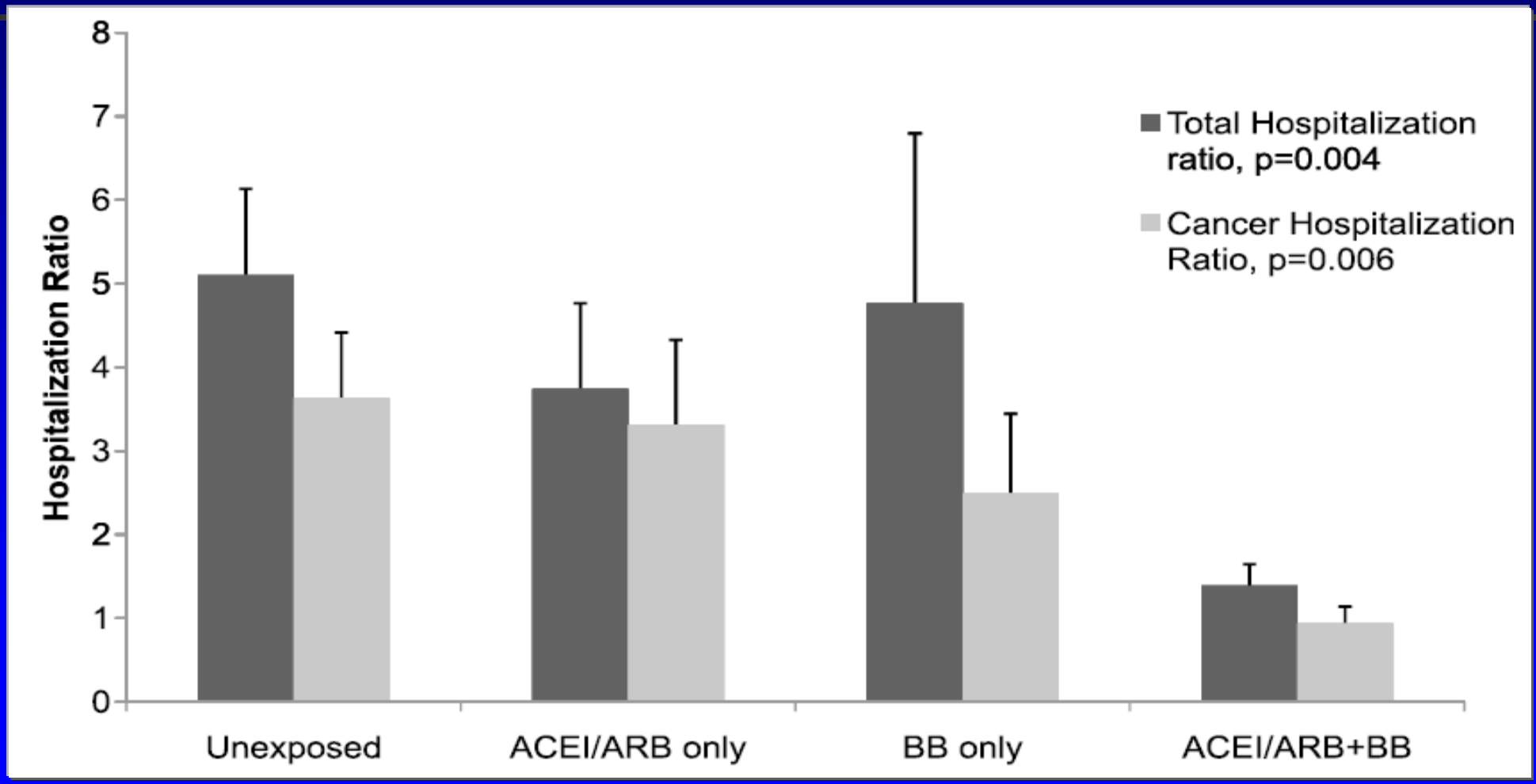
Volume 6 Number 5 October 2013 pp. 539–545 539

Exposure to ACEI/ARB and β-Blockers Is Associated with Improved Survival and Decreased Tumor Progression and Hospitalizations in Patients with Advanced Colon Cancer[1]



ACEI=angiotensin converting enzyme inhibitor; ARB=angiotensin receptor blocker; BB=beta-adrenergic receptor blocker. p value obtained using Chi Square test, comparing ACEI/ARB + BB to unexposed group.

Antihypertensive Therapy and Cancer



survival

tumor progression

Translational Oncology (2013) 6, 539–545

Antihypertensive Therapy and Cancer

IVYSPRING INTERNATIONAL PUBLISHER

Journal of Cancer

2013; 4(7): 549-556. doi: 10.7150/jca.6888

Research Paper

Use of ACE Inhibitors and Angiotensin Receptor Blockers and Primary Breast Cancer Outcomes

Young Kwang Chae[1], Erika N. Brown[2], Xiudong Lei[3], Amal Melhem-Bertrandt[2], Sharon H. Giordano[2], Jennifer K. Litton[2], Gabriel N. Hortobagyi[2], Ana M. Gonzalez-Angulo[2], Mariana Chavez-MacGregor[2]✉

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Antihypertensive Therapy and Cancer

J Clin Hypertens (Greenwich). 2013 Nov 8. doi: 10.1111/jch.12228. [Epub ahead of print]

Lowered Cancer Risk With ACE Inhibitors/ARBs: A Population-Based Cohort Study.

Chiang YY, Chen KB, Tsai TH, Tsai WC.

Author information

Abstract

There are conflicting reports on cancer risk associated with angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs). This retrospective cohort study was conducted to analyze the risk of cancer development in patients who received ACE inhibitors/ARBs as treatment for essential hypertension. Using the Taiwan National Health Insurance Research Database, 297,688 eligible study patients with essential hypertension were identified. According to their antihypertensive prescriptions, the study patients were stratified into an ACE inhibitor group, an ARB group, or a control group. After matching, participants were observed for the occurrence of cancer. In the ACE inhibitor group compared with the control group, the hazard ratio was 0.51 (95% confidence interval, 0.39-0.68). In the ARB group compared with the control group, the hazard ratio was 0.8 (95% confidence interval, 0.65-0.97). Regular use of ACE inhibitors/ARBs was not associated with an increased risk of cancer development and was actually found to decrease overall cancer risk in this study.

Antihypertensive Therapy and Cancer

Cancer Epidemiology
Volume 37, Issue 6, December 2013, Pages 881–885

Antihypertensive medications and survival in patients with cancer: A population-based retrospective cohort study

Signy Holmes[a], E. Jane Griffith[b], Grace Musto[b], Gerald Y. Minuk[a],
[a] Department of Medicine, University of Manitoba, Winnipeg, Canada
[b] CancerCare Manitoba, Winnipeg, Canada

- Provincial Cancer Registry data was linked with a Provincial Drug Program Information Network (DPIN) for patients with lung (*n* = 4241), colorectal (*n* = 3967), breast (*n* = 4019) or prostate (*n* = 3355)
  - BB: no effect
  - ACEi/ARBs use was weakly associated with increased deaths for:
    - breast cancer (HR: 1.22, 95% CI: 1.04–1.44)
    - lung cancer (HR: 1.11, 95% CI: 1.03–1.21)
  - CCB: Increased death in breast cancer (HR: 1.22, 95% CI: 1.02–1.47)
  - Thiazide: There was strong evidence (*p*-value <0.0001) of an increase in deaths colorectal (HR: 1.28, 95% CI: 1.15–1.42), and prostate (HR 1.41, 1.2–1.65) cancer patients.

Antihypertensive Therapy and Cancer (n 1.229.902)

J Hypertens. 2013 Aug;31(8):1669-75. doi: 10.1097/HJH.0b013e3283621ea3.

Angiotensin receptor blockers: are they related to lung cancer?

Rao GA, Mann JR, Shoaibi A, Pai SG, Bottai M, Sutton SS, Haddock KS, Bennett CL, Hebert JR.

	ARBS	No ARBS
Total number	78.075	1.151. 826
A new incident lung cancer		

Chemotherapy and Kidney

Chemotherapy Dose Adjustment

Agent	Dose Adjustment Required when eGFR 10–50 ml/min (%)	Dose Adjustment Required when eGFR <10 ml/min (%)	Evidence Level
Cisplatin	75	50, but avoid if possible	A
Carboplatin	Approximately 50 (AUC-based dose adjustment)	Approximately 25 (AUC-based dose adjustment)	A
Chlorambucil	75	50	D
Ifosfamide	100	75	B
Cyclophosphamide	100	75	B
Daunorubicin	100	100	D
Doxorubicin	100	100	D
Epirubicin	100	100	D
Carmustine	75 for eGFR 30–60 ml/min	Avoid when eGFR <30 ml/min	D
Lomustine	70 for eGFR 30–60 ml/min	Avoid when eGFR <30 ml/min	B
Semustine	70 for eGFR 30–60 ml/min	Avoid when eGFR <30 ml/min	B
Streptozocin	75	50	D
Mitomycin C	100	75	B
Mithramycin	75	50	B
Azacitidine	100	100	B
Gemcitabine	100	100	B
Cytarabine	100	100	D
Methotrexate	50	Avoid	A
Pentostatin	60 for eGFR 30–60 ml/min	Avoid when eGFR <30 ml/min	B
Fludarabine	75	50	D
Cladribine	75	50	D
5-Fluorouracil	100	100	D
Melphalan	75	50	B
Paclitaxel	100	100	A
Vincristine	100	100	B
Vinblastine	100	100	B

Strength of evidence: A, human trials; B, human case studies; C, in vitro data; D, clinical opinion.

Nat Rev Nephrol 5: 450–462, 2009.

Chemotherapies Associated With Kidney Injury

Renal Vasculature

Hemodynamic AKI (capillary leak syndrome)
- IL-2, denileukin diftitox

Thrombotic microangiopathy
- Antiangiogenesis drugs (bevacizumab and tyrosine kinase inhibitors)
- Gemcitabine and cisplatin
- Mitomycin C and IFN

Glomeruli
- Minimal change disease
  - IFN
  - Pamidronate
- FSGS
  - IFN
  - Pamidronate
  - Zoledronate (rare)

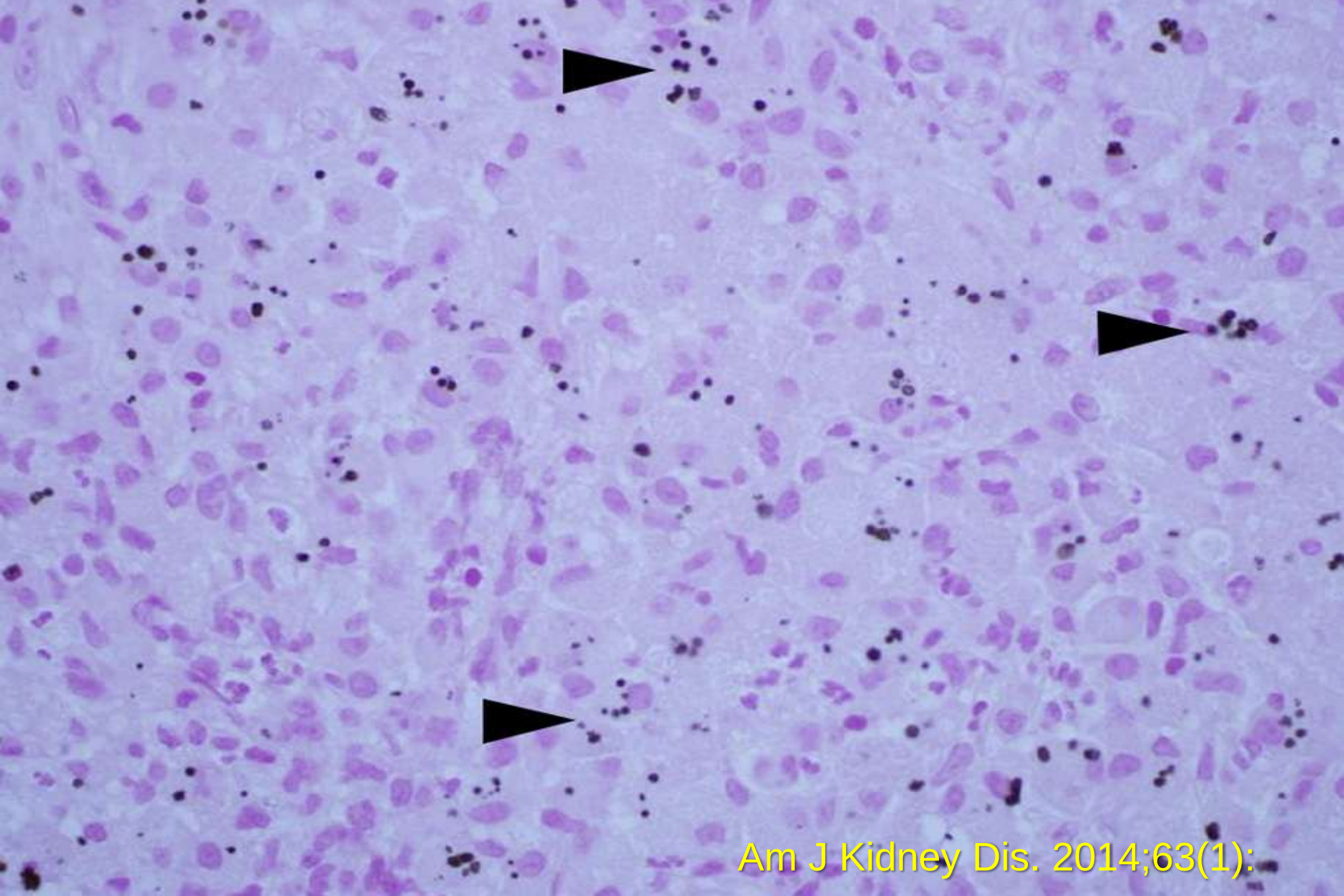
Crystal nephropathy
- Methotrexate

Acute tubular necrosis
- Platinums, zoledronate, ifosfamide, and mithramycin
- Pentostatin, imatinib, diaziquone, and pemetrexed

Tubulopathies
- Fanconi syndrome
  - Cisplatin, ifosfamide, and azacitidine
  - Diaziquone, imatinib, and pemetrexed
- Salt wasting
  - Cisplatin and azacitidine
- Magnesium wasting
  - Cisplatin, cetuximab, and panitumumab
- Nephrogenic diabetes insipidus
  - Cisplatin, ifosfamide, and pemetrexed
- Syndrome of inappropriate antidiuresis
  - Cyclophosphamide and vincristine

Acute interstitial nephritis
- Sorafenib and sunitinib

Clin J Am Soc Nephrol 7: 1713–1721, 2012.



Am J Kidney Dis. 2014;63(1):

Quiz-2

A 65-year-old man presents with the chief complaint of progressive weakness over the past several months. He is normotensive, and his physical examination is unremarkable. Laboratory studies reveal the following: Na 135 mmol/L, Cl 105 mmol/L, K 3.0 mmol/L, HCO_3 18 mEq/L, creatinine 1.8 mg/dl, BUN 22 mg/dl, glucose 110 mg/dl, P_{CO_2} 28 Torr, pH 7.33, hematocrit 25%, white blood cell count 5600/mm^3, and platelets 340,000/mm^3; urinalysis shows trace protein, 1+ glucose, normal sediment, and 24-h urine protein of 4.8 g.

Nephrology Self-Assessment Program - Vol 10, No 2, March 2011